Does parental drinking influence children’s drinking? A systematic review of prospective cohort studies

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ABSTRACT

Aims To evaluate evidence of the capacity for causal inference in studies of associations between parental and offspring alcohol consumption in the general population. Methods A systematic search for, and narrative analysis of, prospective cohort studies of the consequences of drinking, except where assessed prenatally only, or with clinically derived instruments. Primary outcome measures were alcohol use or related problems in offspring, which were collected at least 3 years after exposure measures of parental drinking. The systematic review included 21 studies comprising 26 354 families or parent–child dyads with quantitative effect measures available for each study. Criteria for capacity of causal inference included (1) theory-driven approach and analysis; (2) analytical rigour; and (3) minimization of sources of bias. Results Four of the 21 included studies filled several, but not all, criteria and were assessed to have some capacity for causal inference. These four studies found some evidence that parental drinking predicted drinking behaviour in adolescent offspring. The remaining 17 studies had little or no such capacity. Conclusions There is a fairly large and consistent literature demonstrating that more parental drinking is associated with more drinking in offspring. Despite this, existing evidence is insufficient to warrant causal inferences at this stage.

Keywords Alcohol, causal association, offspring drinking, parental drinking, prospective studies, systematic review.

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INTRODUCTION

Alcohol consumption is one of the major risk factors for loss of healthy years of life globally [1], and in high-income countries it accounts for approximately 19% of disability-adjusted life years (DALYs) and 27% of premature deaths among young people [2]. Assessment of modifiable risk factors for young people’s alcohol consumption and related harms is therefore important. In recent years the scientific and political interest in alcohol’s ‘harm to others’ has grown [3–8], including the possible harms to children from parental drinking. Numerous studies have examined both the possible effects of prenatal alcohol exposure [9,10] and the possible effects on children living with ‘alcoholics’ or parents with serious and long-term alcohol problems [11–13]. However, less is known about how children may be affected by more normative patterns of alcohol consumption and related problems, short of those reaching clinically significant levels, including drinking at lower risk levels and heavy episodic or binge drinking. Previous reviews have addressed associations between parental and offspring drinking behaviour [14,15] and related topics, such as parental supply of alcohol to children [16,17]. Statistically significant associations are very often observed and in many instances they are also interpreted as representing causal effects [14]. However, data may be complex, and associations subject to sources of bias and confounding which may not be measured and controlled. Therefore, careful investigations of the validity of such causal inferences are needed, including thorough assessments of the extent to which other explanations for observed associations can be discounted.

Systematic reviews of prospective cohort studies offer the highest quality observational evidence available for assessment of the true consequences of parental drinking for the onset and development of alcohol use and related problems in young people. Cohort studies have the capacity to ascertain the time order of exposure and outcome and thus
to rule out reverse causality. However, drawing causal inferences from observational epidemiological studies should also be based on testing theory-driven causal hypotheses, applying sufficient analytical rigour and identification and control of sources of bias [18]. The latter includes study design issues such as subject selection and retention, information acquisition and prevention of uncontrolled confounding [19]. In this study we aim to review whether and to what extent prospective cohort studies in the general population provide evidence with capacity for drawing causal inferences on the true effects of parental drinking on their children’s involvement with alcohol.

The importance of assessing possible causal effects of parental drinking pertains not only to a better understanding of complex mechanisms underlying young people’s drinking behaviour, but it has also policy implications. Within a ‘harms to others’ framework, we are interested in the consequences of parental drinking that can be prevented by interventions which reduce parental drinking. In this perspective, both environmental influence and genetic disposition and their interaction are of interest. The literature on familial transmission of alcohol use and of alcohol use disorders (AUD) suggests several mechanisms that may explain observed associations between parental and offspring alcohol use or AUD [20]. These include social learning/modelling effects; parental supply and other forms of physical access to alcohol at home; the mediating role of parenting behaviour; and activation of temperamental predispositions in the presence of environmental stress, the latter being an example of gene × environment interaction [20]. A recent scoping review, which mapped the wider literature [21], identified 99 cohort studies of parental drinking and adverse outcomes in children, and 75 of these analysed drinking behaviour as an outcome. Building on this scoping review, here we review cohort studies of parental and offspring alcohol use in order to: (1) provide an overview of prospective cohort studies estimating parent–offspring drinking associations; (2) assess to what extent these studies have capacity for causal inferences; and (3) examine the strength of the evidence on the size, timing, specificity and probable mechanisms of the effects.

**METHODS**

**Search strategy and selection criteria**

A recent scoping review of cohort studies of parental drinking and adverse outcomes in children [21] provided the basis for more stringent identification of a subset of studies concerned directly with our research questions. The search strategy and selection criteria for this scoping review are described briefly as follows: we searched five electronic databases: MEDLINE; EMBASE; PsycINFO; Global Health; and Web of knowledge, with the last searches being undertaken on 16 October 2013. One author (PK) performed both backward and forward searches to identify any studies that we might have missed [22]. For backward searching we checked the bibliographies of included studies, while for forward searching we used Google Scholar and the Science Citation Index to identify subsequent citations of the included studies. We contacted six experts with a view to identifying additional studies. The database search strategy was devised to include terms across parental alcohol use, children and study design domains.

We sought studies that followed prospectively families or individuals of interest over a period of time, having at least two data collection points. Exposure data collection was required to precede outcome data collection in time. We included studies published in English language peer-reviewed journals from 1980 onwards. Participants included both parents and children from general population samples; those from ‘special populations’ who may have distinct exposure–outcome relationships, e.g. mental health patients, were excluded. We excluded studies where parental drinking was measured with clinical instruments (ICD/DSM) or by brief screening tools derived from diagnostic instruments designed to identify alcohol dependence or ‘alcoholics’ [e.g. ‘The Michigan Alcoholism Screening Test (MAST)’]. Clinical measures were permitted as outcomes. Studies which assessed only alcohol consumption in parents, or consumption plus problems, were included without any lower consumption limits, as were problem measures not derived from ICD/DSM criteria as they were judged probably a priori to assess less severe forms of problems. Studies in which the only parental alcohol data were maternal alcohol use measured during pregnancy were excluded.

A summary of the data collection process is illustrated in the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow-chart (Fig. 1). We followed PRISMA guidance on reporting (Supporting information, Appendix S1) and did not publish a protocol for this study, or include it in a registry. Any form of alcohol outcomes for children were included in this study, and could be assessed at any point in time, including in adulthood. We required a quantitative measure of the size of the effect of parental alcohol use on alcohol outcomes in children, such as odds ratios for binary outcomes or regression or correlation coefficients for outcomes measured on a continuous scale. We also selected studies for this review to include only those that collected exposure data from one or both of the parents, including biological or non-biological parents, as parental reports may be more reliable than offspring’s reports. Indeed, the two correlate, but offspring perceptions underestimate parental drinking [23–26]. We required studies to have a minimum of 3 years between data collection on exposure and outcome, as we wanted...
to capture enduring effects [27]. Finally, we included only those studies that offered a dedicated investigation of the consequences of parental drinking (i.e. not merely inclusion of such a measure as a covariate) and which applied multivariate statistical analyses. Thus, a total of 21 studies were included (Fig. 1). These studies comprised a total of 26 354 families or parent–child dyads.

Quality criteria and data analysis

In the assessment of these 21 studies, we built on contemporary thinking about causal inference in observational studies [18,19,28]. We designated studies as having stronger capacity for causal inference in relation to the aims of this review if the studies had the following characteristics: (1) theory-driven approach and analysis, including suggested mechanisms of effects, and identification of important confounding factors; (2) analytical rigour including adequate analyses to assess suggested mechanism(s), assessment of possible interactions between maternal and paternal drinking, and taking account of probable confounding factors by extent of adjustments in multivariate models; and (3) minimization of sources of bias, including having data on both parents’ drinking and collected separately, exposure data collected at ages at which it could plausibly influence offspring drinking.
(i.e. in childhood or adolescence); a graded exposure measure in order to obtain an indication of a dose–response relationship; and sufficient statistical power to reduce Type II error risk. Regarding the theory-driven approach, we assumed that if there is a causal effect of parental drinking on that of their children, it is likely that both parents’ drinking behaviour are relevant. Therefore, we considered both parents’ drinking behaviour and their additive or interactive effects to be of interest. These would preferably be self-reported separately, and modelled to obtain additive/interactive effects. Presence of the theory-driven approach, including suggested mechanisms and identification of important confounders, is a logical prerequisite for analytical rigour. Therefore, adjustment for a larger number of variables (e.g. maternal smoking) in the analyses does not necessarily imply better control for important confounding factors. Finally, in sensitivity analyses we assessed whether or to what extent our inclusion criteria for this review affected the main results. We summarized the outcomes of studies in the scoping review that would meet other candidate inclusion criteria for this study (e.g. having a less than 3-year gap between exposure and outcome, or child report of parental drinking) and compared these data to the outcomes of the 21 selected studies.

**RESULTS**

The studies were conducted in six different countries: the United States (n = 11) [29–39; Australia (n = 3) [40–42, the Netherlands (n = 3) [43–45]; New Zealand (n = 2) [46,47]; Finland (n = 1) [48; and the United Kingdom (n = 1)]49). Multiple study reports were based on the same cohorts; altogether 16 distinct cohorts were identified. For each of the 21 studies, in Table 1 we have presented the study characteristics for cohort type, sample size including attrition, exposure and outcome measures and main findings, and assessed capacity for causal inference in Table 2.

The exposure measure varied substantially between the studies with regard to type of drinking behaviour (e.g. drinking frequency, typical weekly volume), age of exposure and putative relationship to outcomes (from before pregnancy to young adulthood), and whose drinking behaviour was measured (only mother, only father, separate measures for both parents or combined measure for both parents; Table 1).

The outcome was one or several measures of drinking behaviour (e.g. drinking frequency, early onset of drinking or heavy episodic drinking frequency) in 16 of the studies. In five studies the outcome was some kind of alcohol-related problem (e.g. alcohol dependence), either as a single outcome (three studies) [35,40,45] or in addition to a measure of drinking behaviour (two studies) [36,43]. In 13 of the studies the outcome measures were obtained only or mainly during the teenage years, whereas in seven studies the outcome measures were obtained mainly or only in young adulthood [30,35,39,40,44–46], and in one study at the age of 10 years [49]. In light of observed heterogeneity and the consequent lack of data appropriate for meta-analysis, we undertook a narrative synthesis of included study findings and risk of bias.

The vast majority (19 of 21 studies) reported at least one positive association between parental drinking and offspring’s alcohol-related outcome, while only two studies [31,47] found no statistically significant association. This pattern held for both adolescent and young adult outcomes (Table 1). Of eight studies that examined mother’s and father’s possible drinking consequences separately, three studies reported that both parents’ drinking behaviour predicted that of the child [33,39,42], three studies found that only mother’s drinking predicted the outcome [44,46,49], and two studies found that only father’s drinking predicted the outcome [43,45] (Table 1). Among four studies addressing same sex versus opposite sex associations between parent and offspring drinking [39,42,45,46], the findings were mixed (Table 1).

Next, we assessed the studies’ capacity for causal inference according to the aims of this study and the evaluation framework described previously in relation to parental drinking and alcohol-related outcomes in offspring. All studies had some favourable characteristics in this respect: for instance, graded exposure measures or large sample sizes (Table 2). However, the majority of the studies were not well designed to evaluate possible causation and lacked an explicit theoretical conceptualization of their research aims. In fact, none of the studies identified and accounted for theory-driven important confounding factors in order to interrogate observed associations. Therefore, we found that none of the 21 studies could be considered as having strong capacity for causal inference. Four studies [37,42,43,48] were found to have some inferential capacity in this respect and the remaining 17 studies had little or no such capacity (see Table 2 for a summary of the basis of categorization of each included study).

Among the four studies [37,42,43,48] with some capacity for causal inference, all found some evidence that parental drinking predicted drinking behaviour in offspring (Table 3). Three of these studies had clear theory-driven analyses of the association between parental and offspring drinking [37,43,48]. They examined specific mediation mechanisms, assuming that the association between parental and offspring drinking was mediated by either parenting practices [48], by alcohol-specific communication [43] or by poor inhibitory control in offspring [37]. Conversely, the study by Alati and co-workers [42] accounted for some theory-driven covariates in the analyses, but not within a clear framework of testing causal mechanisms,
<table>
<thead>
<tr>
<th>Study</th>
<th>Characteristics</th>
<th>Exposure measure</th>
<th>Outcome(s) measure</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>First author, year, reference</td>
<td>Sample type and size</td>
<td>Follow-up rate (%)</td>
<td>Type</td>
<td>Child's age</td>
</tr>
<tr>
<td>Alati, 2005 [40]</td>
<td>Birth cohort, n = 2386</td>
<td>35a</td>
<td>Drinking frequency</td>
<td>None</td>
</tr>
<tr>
<td>Alati, 2008 [41]</td>
<td>Birth cohort, n = 4363</td>
<td>60b</td>
<td>Usual quantity 3+</td>
<td>None</td>
</tr>
<tr>
<td>Alati, 2014 [42]</td>
<td>Birth cohort, n = 751</td>
<td>53b</td>
<td>Drinking categories</td>
<td>None</td>
</tr>
<tr>
<td>Armstrong, 2013 [29]</td>
<td>Community sample, n = 374</td>
<td>66b</td>
<td>Usual quantity</td>
<td>None</td>
</tr>
<tr>
<td>Bailey, 2006 [30]</td>
<td>Community sample, n = 208</td>
<td>55b</td>
<td>Binge drinking (5+) frequency</td>
<td>None</td>
</tr>
<tr>
<td>Burk, 2011 [31]</td>
<td>Community sample, n = 362</td>
<td>67b</td>
<td>Quantity per day</td>
<td>None</td>
</tr>
<tr>
<td>Casswell, 2002 [46]</td>
<td>Birth cohort, n = 714</td>
<td>77b</td>
<td>Drinking frequency and quantity</td>
<td>None</td>
</tr>
</tbody>
</table>
Cortes, 2009 [32] School students, \(n = 792\)  
Alcohol use, not specified  
Past 30 days  
Mother only  
At ages 8–12  
Alcohol use frequency growth  
At ages 13–17  
Maternal alcohol use predicted growth in child alcohol use (\(\beta = 0.10, P < 0.05\))  
Yes

Donovan, 2011 [33] Community sample, \(n = 393\)  
Drinking frequency  
Past 6 months  
Both parents combined  
At age 10  
Age at drinking initiation, early onset  
Before age 15  
Average parental drinking frequency predicted early onset of drinking (OR = 1.007, \(P < 0.05\))  
Yes

Duncan, 2011 [34] Community sample, \(n = 256\)  
Drinking frequency  
None  
9  
Both parents combined  
Ages 13, 15  
Drinking frequency  
Ages 18, 20  
More frequent parental drinking predicted increased youth drinking over time (\(\beta = 0.10, P < 0.05\))  
Yes

Fergusson, 1995 [47] Birth cohort, \(n = 953\)  
Typical weekly volume  
Both parents combined  
Age 11  
Amount/ occasion, hazardous drinking  
Ages 14, 16  
Parental drinking did not predict drinking at age 14, and was not directly associated with hazardous drinking at age 16  
No (at 14) and yes (at 16)

Guo, 2001 [35] School students, \(n = 808\)  
Alcohol use, not specified  
None  
Both parents combined  
Ages 10, 14, 16  
Alcohol abuse/ dependence  
Age 21  
Only parental alcohol use at age 16 predicted alcohol abuse (OR = 1.42, \(P < 0.01\)) and alcohol dependence (OR = 1.65, \(P < 0.01\))  
Yes

Hawkins, 1997 [36] Students, \(n = 757\)  
Drinking frequency  
None  
Both parents combined  
Ages 10–11  
Alcohol initiation, alcohol misuse  
Ages 17–18  
Parental drinking predicted earlier drinking initiation (\(\beta = -0.19, P < 0.05\)), no direct association with alcohol misuse at 17–18  
Yes

Latendresse, 2008 [48] Cohort twins, \(n = 4731\)  
Drinking frequency, intoxication freq  
Current 9  
Both parents combined  
Ages 11–12  
Drinking behaviour  
Ages 14 and 17.5  
Parental drinking behaviours predicted child's alcohol use and intoxication at ages 14 and 17 (8 path coefficients, range 0.02–0.16, \(P < 0.001\) for all)  
Yes

Macleod, 2008 [49] Birth cohort, \(n = 4064\)  
Parental drinking, None 3 cat,  
Both parents separate  
Ages 0–4  
Alcohol use  
Age 10  
Maternal drinking predicted only alcohol use (OR = 2.6, \(P < 0.01\)), no association with paternal drinking  
Yes

Mares, 2011 [43] Families, \(n = 428\)  
Drinking frequency, volume  
Past 4–6 weeks (frequency)  
Both parents separate  
Ages 13–16  
Excessive alcohol use, related problems  
Ages 17–20  
Paternal, but not maternal, drinking predicted only excessive drinking (\(\beta = 0.16\) for older and \(\beta = 0.17\) for younger adolescents, \(P < 0.05\))  
Yes
<table>
<thead>
<tr>
<th>Study</th>
<th>Characteristics</th>
<th>Exposure measure</th>
<th>Outcome(s) measure</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pears, 2007</td>
<td>103 families</td>
<td>Drinking frequency</td>
<td>Both parents combined</td>
<td>Ages 9–10 Alcohol use frequency Ages 16–18 Grandparents' alcohol use predicted parents' alcohol use (path coefficient = 0.22, P &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>68^b</td>
<td>None</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poelen, 2007</td>
<td>Twin families,</td>
<td>Drinking frequency</td>
<td>Both parents</td>
<td>Ages 16–18 Grandparents' alcohol use predicted parents' alcohol use (path coefficient = 0.22, P &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>n = 1779</td>
<td>None 3</td>
<td>separate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>47^b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poelen, 2009</td>
<td>Twin families,</td>
<td>Drinking frequency</td>
<td>Both parents</td>
<td>Ages 19–32 Only paternal drinking a few times/week predicted problem drinking 7 years later (OR = 1.78, P &lt; 0.001)</td>
</tr>
<tr>
<td></td>
<td>n = 1796</td>
<td>None 3</td>
<td>separate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>48^b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tyler, 2006</td>
<td>Youth cohort,</td>
<td>Binge drinking</td>
<td>Mother only</td>
<td>Ages 14–16 Mother's binge drinking predicted binge drinking at ages 14–16 (β = 0.171, P &lt; 0.01), not at ages 16–18</td>
</tr>
<tr>
<td></td>
<td>n = 244</td>
<td>(5+) Past 30 2</td>
<td>Ages 10–12 Binge</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>days</td>
<td>drinking (5+)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Webster, 1989</td>
<td>Community sample,</td>
<td>Volume</td>
<td>Both parents</td>
<td>Mean ages Father's drinking (partial r = 0.15), P = 0.05 and mother's drinking (partial r = 0.16, P = 0.04) predicted alcohol use in sons, only father's drinking (partial r = 0.29, P &lt; 0.001) predicted alcohol use in daughters</td>
</tr>
<tr>
<td></td>
<td>Not clear</td>
<td>None</td>
<td>separate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n = 420 families</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Sample size is the number of people in the (multivariate) analysis. Proportion that was followed-up and completed Composite International Diagnostic Interview (CIDI); our calculation based on the figures in the article; net sample as proportion of initial gross sample when missing data excluded. TF = time-frame; cat = number of categories; parents comb = measure of parental drinking combined; CAGE = Cut-down, Annoyed, Guilt, Eye-opener; OR = odds ratio; SD = standard deviation.
<table>
<thead>
<tr>
<th>Author, year reference</th>
<th>Main focus on parent—offspring drinking association</th>
<th>Theory-driven analyses aimed at assessing causality</th>
<th>Identification of important confounding factors</th>
<th>Sample size</th>
<th>Exposure measure graded</th>
<th>Relevant period</th>
<th>Assessment of parental drinking interaction</th>
<th>Notes on outcome measure</th>
<th>Capacity for causal inference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alati, 2005 [40]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Large</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Little</td>
</tr>
<tr>
<td>Alati, 2008 [41]</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Large</td>
<td>No</td>
<td>No</td>
<td>No, long before outcome</td>
<td>No</td>
<td>Little</td>
</tr>
<tr>
<td>Alati, 2014 [42]</td>
<td>Yes</td>
<td>Suggests parenting may mediate the association. Analysis not clearly aimed at addressing causality</td>
<td>Time-varying covariates included</td>
<td>Acceptable</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Limited data on key measure</td>
<td>Some</td>
</tr>
<tr>
<td>Armstrong 2013 [29]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>On the smaller side</td>
<td>Yes</td>
<td>Somewhat early</td>
<td>No</td>
<td>No</td>
<td>Little</td>
</tr>
<tr>
<td>Bailey, 2006 [30]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>On the smaller side</td>
<td>Small</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Little</td>
</tr>
<tr>
<td>Burk, 2011 [31]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>On the smaller side</td>
<td>Acceptable</td>
<td>No</td>
<td>Many years from exposure to outcome</td>
<td>No</td>
<td>Trajectories from ages 18–26</td>
</tr>
<tr>
<td>Casswell, 2002 [46]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Acceptable</td>
<td>Vaguely described</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Little</td>
</tr>
<tr>
<td>Cortes, 2009 [32]</td>
<td>No</td>
<td>No</td>
<td>Unclear</td>
<td>Acceptable</td>
<td>Vaguely described</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Little</td>
</tr>
<tr>
<td>Donovan, 2011 [33]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>On the smaller side</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Validation of outcome measure</td>
<td>Little</td>
</tr>
<tr>
<td>Duncan, 2011 [34]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Small</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Crude</td>
</tr>
<tr>
<td>Fergusson, 1995 [47]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Acceptable</td>
<td>Not described</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Additive effect implied</td>
</tr>
<tr>
<td>Guo, 2001 [35]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Acceptable</td>
<td>Vaguely described</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Construction and validity not clear</td>
</tr>
<tr>
<td>Hawkins, 1997 [36]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Acceptable</td>
<td>Vaguely described</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Two aspects (similar for parents and children) modelled</td>
</tr>
<tr>
<td>Latendresse, 2008 [48]</td>
<td>Yes</td>
<td>Suggested mediation mechanisms examined</td>
<td>3 covariates included, probably lacks important confounding factors</td>
<td>Large</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Some</td>
</tr>
</tbody>
</table>

(Continues)
Table 2. (Continued)

<table>
<thead>
<tr>
<th>Author, year, reference</th>
<th>Main focus on parent–offspring drinking association</th>
<th>Theory-driven analyses aimed at assessing causality</th>
<th>Identification of important confounding factors</th>
<th>Sample size</th>
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<th>Relevant period</th>
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<th>Notes on outcome measure</th>
<th>Capacity for causal inference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macleod, 2008 [49]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Large</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Low-prevalent outcome and substantial missing data</td>
<td>Little</td>
</tr>
<tr>
<td>Mares, 2011 [43]</td>
<td>Yes</td>
<td>Suggested modelling effects via alcohol communication</td>
<td>No</td>
<td>On the lower side</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Some</td>
<td></td>
</tr>
<tr>
<td>Pears, 2007 [37]</td>
<td>Yes</td>
<td>Suggested indirect effects through parental discipline and offspring's inhibitory control</td>
<td>No</td>
<td>Very small</td>
<td>Vaguely described</td>
<td>Yes</td>
<td>No</td>
<td>Adjacent survey years combined</td>
<td>Some</td>
</tr>
<tr>
<td>Poelen, 2007 [44]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Large</td>
<td>Yes</td>
<td>Partly</td>
<td>No</td>
<td>Little</td>
<td></td>
</tr>
<tr>
<td>Poelen, 2009 [45]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Large</td>
<td>Yes</td>
<td>Partly</td>
<td>No</td>
<td>Little</td>
<td></td>
</tr>
<tr>
<td>Tyler, 2006 [38]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Small</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Little</td>
<td></td>
</tr>
<tr>
<td>Webster, 1989 [39]</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Small</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Little</td>
<td></td>
</tr>
</tbody>
</table>
thereby hampering substantive interpretation of the reported findings.

The study by Mares and co-workers [43] found direct effects of paternal, but not maternal drinking; however, the apparent differential effects may be due to insufficient statistical power and model misspecification (intercorrelated measures of maternal and paternal drinking were estimated simultaneously). The findings also, in part, indicated indirect effects of parental alcohol-related problems through parental–child communication: more alcohol-related problems in parents predicted more alcohol-specific communication, which in turn was associated with less excessive drinking (β = -0.14) and less alcohol-related problems (β = -0.13) in offspring in indirect path models.

The study by Latendresse and co-workers [48] is particularly noteworthy in the context of our research aims. The authors found that the association between parental (probably mainly paternal) and offspring drinking was mediated in part by parental monitoring and discipline; more so at age 14. As hypothesized, both effects mediated partly by parental monitoring and discipline; more so at age 14. Gender, family structure, and zygosity were included as co-variates in multiple mediation models.

The study by Pears and co-workers [37] did not find any mediation effect of poor inhibitory control in offspring, which may well be due to insufficient statistical power, or there may be no such effect.
that if there is a causal and partly mediated effect of parental drinking it may be an additive or interaction effect of both parents' drinking behaviour, which was not addressed in this study, or in any of the other studies included in this review. Consequently, findings on the size, timing, specificity and probable mechanisms of the effects are very limited across these studies.

Finally, we assessed whether our inclusion criteria for this analytical review had impacted upon our findings. The 21 studies included in our review differed very little from the findings of 28 excluded studies with either child report of exposure, or a gap of less than 3 years between exposure and outcome measurement. A combined measure for both parents tended to be used when parental drinking was reported by offspring, and only 55% of these studies found an association between parental and offspring drinking, compared to 78% of studies using parental report ($\chi^2 = 3.51, P = 0.06$).

**DISCUSSION**

This study is the first systematic review of cohort studies which interrogates the basis for causal inference on the effects of parental drinking on children’s alcohol outcomes. It has demonstrated that among the many prospective cohort studies that have addressed whether and to what extent parental drinking predicts drinking behaviour in offspring, few have been designed to measure validly the effects of parental drinking on the drinking of their offspring. Almost all prospective studies on this topic have found that parental drinking predicts drinking behaviour in their children; that is, when one or both parents drink more, their offspring are more likely to report more drinking or more alcohol-related problems later on than others in the cohort. Findings on the relative effects of paternal versus maternal drinking are different in the studies by Alati [42] and Mares [43], with maternal drinking more important in the former and paternal drinking more so in the latter. This overall consistency in findings is, however, not sufficient by itself to indicate a causal relationship [28]. The somewhat mixed findings regarding the differential impacts of maternal and paternal drinking, the sparse use of theory-driven analyses, and thus the lack of identification and control for relevant confounding factors, the small data sets and consequent limited capacity for detecting associations of moderate magnitude, are all factors that imply caution is warranted about the consequences of parental drinking under consideration.

Closely related to the topic of this review is the literature on familial transmission of alcohol misuse. Relying on both twin and adoption studies this literature suggests that genetic predisposition and interactions between genes and environment are important [50]. A striking observation, therefore, is that studies included in this review, and particularly studies using twin data [44,45,48], did not address these factors. Possible mechanisms that were suggested and examined in some studies in this review [43,48,51] were all in the behavioural domain. Thus, it seems that data from designs other than prospective cohort studies are more informed by genetic data and gene–environment interactions, and it remains to be seen how far rigorously designed systematic reviews may alter evidential claims about genetic heritability in a wider range of study designs.

The four studies with some capacity for causal inference all found that parental drinking predicted greater involvement in drinking in offspring. However, the possibility that these observed associations are spurious needs consideration. Some possible sources of spurious associations are as follows: (a) common local environment (neighbourhood, community) influences on both parental and offspring drinking, such as physical access to alcohol and price; (b) common cultural or religious factors including both those that enhance and limit or proscribe drinking that affect both parental and offspring drinking; and (c) parental comorbidity/temperament and other psychobiological factors affected by genetic transmission. These factors may either moderate or mediate mechanisms, or both, and are seldom addressed in the studies included in this review. Failure to demonstrate mediation effects as they were hypothesized in two [37,43] of three studies [37,43,48], all of which were assessed to have some capacity for causal inference, and some inconsistency regarding possible effects of maternal drinking, may well be due to insufficient statistical power and model misspecification, meaning that the hypothesized mediation effects and specific effects of maternal drinking should not be discarded from further investigations.

As well as evaluation of the included studies, consideration of the strengths and limitations of this study is appropriate. A fairly large literature on cohort studies of parental and offspring drinking was identified through extensive and systematic literature searches. Applying a set of criteria for drawing causal inferences, including theoretical underpinning and analytical rigour, enhanced the systematic evaluation of the studies' contributions in this respect. This process has also been made as transparently as possible, permitting readers to assess its rigour and its limitations. Studies from different national and cultural contexts were identified, although these were restricted entirely to Anglophone and northern European countries. The selection of studies was restricted to those published in the English language, implying that relevant studies in other languages may exist, but have not been identified in this review. Diverse measures of exposures and outcomes entailed difficulties in conducting quantitative syntheses and it may have been possible to pursue quantitative investigations, notwithstanding the heterogeneity we encountered.
The lack of standardization in measurement may also be regarded as a limitation of the literature as a whole, hampering comparability across studies. For instance, with respect to adolescents’ drinking behaviour, there is a distinction between sipping and consumption of full beverages [52], and relatedly it is worth considering that age of onset/initiation as an outcome measure (as in [33,36,49]) may have a distinct relationship to parental drinking. It may also have different consequences from other alcohol involvement outcomes investigated here, which may also be heterogeneous in this regard, and the need for an intergenerational life-course perspective should be considered [53].

The determination of study quality did not consider self-report bias in both exposure and outcome measures, and is otherwise absent from this study design except in separating the two reports in time. Self-reported drinking behaviour is often under-reported, and this leads to a biased estimate of the associations with consequences [27]. The possibility of publication bias needs also to be considered [54]. As null-findings are less likely to be published, the observed associations in the vast majority of studies included here may represent an exaggeration of the true picture. Due to the nature of the literature, we have not been able to assess this quantitatively. Finally, our study findings need to be interpreted within the context of the emphasis we have placed on the testing of theory-driven causal hypotheses and other aspects of the design of this systematic review.

Strategies to prevent harmful drinking in young people and its acute and long-term health and social consequences may target parents and parental drinking and include general population strategies [55,56] and specific parent-targeted programmes [57,58], but the effectiveness of the latter is contingent upon an underlying causal effect of parental drinking on that of their children. The findings from studies with some capacity for causal inference suggest that such effects may actually exist. This study has demonstrated that there is currently little strong evidence, however, of a causal effect of parental drinking on that of their children. More well-designed theory-driven cohort studies addressing the possible influence of parental drinking on that of their children, as well as other putative risk factors, are needed urgently in order to understand more clearly the true burden of alcohol’s harm to others, and to determine the most appropriate ways to prevent intergenerational alcohol problems.

Declaration of interest
None.

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References


**Supporting Information**

Additional supporting information may be found in the online version of this article at the publisher’s web-site.